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CLINICAL MANIFESTATION OF ZINC PHOSPHIDE POISONING OF ANIMALS
[Klinicheskoye proyavleniye otravleniya zhivotnykh fosfidom tsinka]

by

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To observe the clinical picture of zinc phosphide poisoning, 120 mammals (pigs, sheep, goats, calves, rabbits, dogs and cats) were administered a zinc phosphide preparation in doses of 10-150 mg/kg. In most cases the symptoms of poisoning developed rapidly, culminating in a fatal outcome in a matter of hours, and signs of poisoning were similar in all species of experimental animal used. The animals first experienced short-term stimulation, manifested by unrest, whining, dashing or leaping about the cage etc, followed by depression giving way to paralysis of the extremities. Total loss of appetite, vomiting (in pigs, cats and dogs) arythmia of respiration, tachycardia, bradychardia, hypothermia and a terminal comatose state gave further evidence of zinc oxide's injurious effect upon the animals' nervous systems.

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The ever broader employment of various toxic chemicals in the national economy creates conditions for animals' contact with them, which frequently leads to mass poisoning. One such toxic chemical is zinc phosphide, which pertains to the zoocides with an acute effect.

According to our data, 31.5% of all poisonings of mammals and birds reported on the farms of Northeast Kazakhstan during 1956-1965 were due to zinc phosphide. The majority of cases of such poisonings were noted from April to October and very rarely in December. This is connected with the time during which zinc phosphide is used for disinfestation purposes and with the pasturing period.

In connection with the insufficient degree to which the clinical picture has been studied in zinc phosphide poisoning and the contradictory data on this question in the literature, we set ourselves the goal of tracing the development of the symptoms of this poisoning in an experiment. We used 120 mammals (10 pigs, 10 sheep, 12 goats, 4 calves, 35 rabbits, 29 dogs, 20 cats) in the experiments. We tried various doses (from 10 to 150 mg/kg) of zinc phosphide on them. We administered zinc phosphide individually in the form of an aqueous suspension using an oral-esophagal catheter or we gave it to them with their feed. We employed the preparation several times each on some carnivores and rabbits. We produced lethal poisoning in all animals.

In the majority of cases, the course of experimental poisoning of animals was acute and lasted several hours; its symptoms

increased rapidly. For example, in the four-month-old heifer No. 3534 with an average of 70 kg of fat, to which we gave 7 g of zinc phosphide on an empty stomach (a dose of 100 mg/kg), the clinical picture developed in the following way. The overall condition was within normal bounds during the first hour and a half. Then we observed some unrest: the animal lay down, stood up and went from place to place. Slight unsteadiness was noticeable in movement. The body temperature increased by 0.5° as compared with the original.

After two hours the animal was standing with its extremities planted apart and strained; the animal licked itself, opened its mouth (as if yawning), stretched itself, looked at its abdomen, licked its skin and changed the position of its head (downwards, toward its spine, backwards etc). After three hours the heifer lay down, extended its neck and from time to time a muscular tremor was noted in the region of its neck and shoulder girdle. The body temperature became 1.2° higher than the original. In the next hour, unrest was again noted in the heifer and short-term muscular tremor of the whole body appeared from time to time. The animal moved with difficulty (halting and unsteady gait).

Sharp depression developed in the animal after five hours. It stood with difficulty, flinched sharply when the skin was touched and short-term spasms were periodically noted; the body temperature rose to 39.7°. The heifer lay down, extending the pelvic extremities and tucking the thoracic ones under, made attempts to rise, could not stand independently after being

raised up, and fell. The muscular tremor was repeated more often; the respiration became heavy and arythmic. We killed the animal when it was close to its death throes (six hours after administration of zinc phosphide).

We noted approximately the same clinical picture in a six-month-old bull calf to which we administered the preparation in a dose of 60 mg/kg. However the symptoms of poisoning developed somewhat later.

In goats and sheep, the clinical signs coincided to a certain extent with those in cattle.

We did not note changes in the general condition of pigs poisoned with the preparation in doses of 60 and 100 mg/kg during the first hour and a half; then signs of unrest developed. Two hours after they took morning feed, we observed multiple vomiting in all of the animals; it also occurred at a subsequent time. An odor of hydrogen phosphide was detected in the first batches of vomit. In the animals poisoned by the preparation in a dose of 100 mg/kg, weakness appeared 1-2 hours after vomiting, the animals hid in a corner and lay down and could not stand up or stand independently. Spasms were noted at the same time in 1 out of 3 young pigs in this group. Signs of depression appeared later after vomiting in 2 pigs to whom we had administered zinc phosphide in a dose of 60 mg/kg.

In dogs to whom we fed zinc phosphide with sausage meat or bread, signs of unrest sometimes appeared after 30-40 minutes: they ran, yelped, drank water and then multiple vomiting occurred;

if vomiting appeared in the first two hours after taking feed only slight depression, which passed after a day, was subsequently observed in the dogs. If vomiting took place later, then poisoning culminating in death developed in the animals in the majority of cases. For example, in puppy No. 3019, to whom we fed zinc phosphide in a dose of 40 mg/kg with sausage meat, we noted vomiting after three hours and it was repeated 3 times in 10 minutes. Then signs of depression appeared in the dog (it hid in a corner, lay unmoving and did not answer a shout). Depression grew sharply stronger 18 hours after priming (it lay in an unnatural position with the head turned 180 degrees backwards, at times executed swimming movements, tried to stand; the hind legs dragged when moving and it fell). It had periods of vomiting with deposit of a small quantity of a gray, viscous, frothy substance with an odor of hydrogen phosphide; appetite and desire for water were absent. It died 28 hours from the moment of priming in a state of sharp depression and general weakness.

Three dogs primed with the preparation in doses of 30 and 60 mg/kg died with similar clinical signs; however they developed more rapidly. The majority of the other dogs poisoned by the same high doses of zinc phosphide remained alive thanks to rapidly occurring vomiting and elimination of the toxin from the stomach.

Cats were uneasy and meowed after taking poisoned feed. Then vomiting and salivation occurred in them, they hid in the corner of the cage, sat unmoving, did not react to stimuli and died

amidst increasing signs of depression.

Unrest was also noted in rabbits after zinc phosphide poisoning (they rushed forward, gave cries, and flinched sharply when their skin was touched). Sometimes we observed short-term spasms in them prior to death. Thus rabbit No. 3136, poisoned by the preparation in a dose of 20 mg/kg, ate hay and drank water for three hours. Then unrest occurred (the rabbit sprang about the cage, rushed forward irrepressibly and thrust itself against the walls and netting of the cage). After a brief period of stimulation, signs of depression appeared in it. After six hours depression was sharp, the rabbit layed in an unnatural position, made disordered and swimming movements, tried to rise but could not, and then spasms occurred in it. At this time respiration was deep, 54 times per minute; the pulse, 84, arythmic; and body temperature, 34.5°. The rabbit died eight hours after priming amidst phenomena of general weakness.

Therefore the clinical signs of zinc phosphide poisoning were to some degree similar in mammals. They testify to predominant injury to the central nervous system. We first observed signs of stimulation manifested by unrest, rushing forward and a feeling of fear. Depression set in after short-term stimulation; beginning with somnolence, it turned into sopor. In the depression phase the illness manifested itself through weakening of reflexes and decrease in deep sensitivity, in consequence of which ataxia of a static nature that was intensified in walking revealed itself. In depression, the animals as a rule are

forced to assume a prone position, swimming motions being preserved at first with paralysis of the paraplegic type then developing.

Disturbance of the function of other organs develops consequent to disruption of the activity of the nervous system. Decrease in appetite up to complete disappearance is noted; vomiting is always observed in pigs, dogs and cats. Disturbances on the part of the organs of respiration are manifested mainly by arythmia of respiratory movements.

Tachycardia and intermittant arythmia of the heart is observed in disturbance of the function of the circulatory organs. The premortal signs: a comatose state, paralysis of the extremities, bradycardia, oligopneic respiration and hypothermia.